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(ROSPATENT) added to list of core patent offices covered
NEWS 4 FEB 28 PATDPAFULL - New display fields provide for legal status
data from INPADOC
NEWS 5 FEB 28 BABS - Current-awareness alerts (SDIs) available
NEWS 6 FEB 28 MEDLINE/LMEDLINE reloaded
NEWS 7 MAR 02 GBFULL: New full-text patent database on STN
NEWS 8 MAR 03 REGISTRY/ZREGISTRY - Sequence annotations enhanced
NEWS 9 MAR 03 MEDLINE file segment of TOXCENTER reloaded
NEWS 10 MAR 22 KOREAPAT now updated monthly; patent information enhanced
NEWS 11 MAR 22 Original IDE display format returns to REGISTRY/ZREGISTRY
NEWS 12 MAR 22 PATDPASPC - New patent database available
NEWS 13 MAR 22 REGISTRY/ZREGISTRY enhanced with experimental property tags

NEWS EXPRESS JANUARY 10 CURRENT WINDOWS VERSION IS V7.01a, CURRENT
MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
AND CURRENT DISCOVER FILE IS DATED 10 JANUARY 2005

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FILE 'HOME' ENTERED AT 17:28:20 ON 25 MAR 2005

=> index bioscience

FILE 'DRUGMONOG' ACCESS NOT AUTHORIZED

COST IN U.S. DOLLARS

SINCE FILE	TOTAL
ENTRY	SESSION
0.21	0.21

FULL ESTIMATED COST

INDEX 'ADISCTI, ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, ANTE, AQUALINE,
AQUASCI, BIOBUSINESS, BIOCOMMERCE, BIOENG, BIOSIS, BIOTECHABS, BIOTECHDS,

BIOTECHNO, CABA, CANCERLIT, CAPLUS, CEABA-VTB, CEN, CIN, CONFSCI, CROPB,
CROPU, DDFB, DDFU, DGENE, DISSABS, ...' ENTERED AT 17:28:37 ON 25 MAR 2005

75 FILES IN THE FILE LIST IN STNINDEX

Enter SET DETAIL ON to see search term postings or to view
search error messages that display as 0* with SET DETAIL OFF.

=> "cholera toxin B sub-unit" and "heat shock protein 60"

13 FILES SEARCHED...
17 FILES SEARCHED...
27 FILES SEARCHED...
34 FILES SEARCHED...
48 FILES SEARCHED...
55 FILES SEARCHED...
68 FILES SEARCHED...
73 FILES SEARCHED...

0 FILES HAVE ONE OR MORE ANSWERS, 75 FILES SEARCHED IN STNINDEX

L1 QUE "CHOLERA TOXIN B SUB-UNIT" AND "HEAT SHOCK PROTEIN 60"

=> "behcet's disease"

1 FILE ADISCTI
4 FILE ADISNEWS
454 FILE BIOSIS
2 FILE BIOTECHABS
2 FILE BIOTECHDS
1 FILE BIOTECHNO
1 FILE CABA
16 FILES SEARCHED...
2 FILE CANCERLIT
63 FILE CAPLUS
2 FILE CONFSCI
32 FILE DDFB
15 FILE DDFU
5 FILE DGENE
27 FILES SEARCHED...
32 FILE DRUGB
21 FILE DRUGU
1 FILE EMBAL
21 FILE EMBASE
2 FILE ESBIODASE
4 FILE GENBANK
14 FILE JICST-EPLUS
1 FILE LIFESCI
10 FILE MEDICONF
21 FILE MEDLINE
50 FILES SEARCHED...
6 FILE PASCAL
1643 FILE SCISEARCH
64 FILE TOXCENTER
17 FILE USPATFULL
70 FILES SEARCHED...
15 FILE WPIDS
15 FILE WPINDEX

29 FILES HAVE ONE OR MORE ANSWERS, 75 FILES SEARCHED IN STNINDEX

L2 QUE "BEHCET'S DISEASE"

=> 12 rCTB

MISSING OPERATOR L2 RCTB

The search profile that was entered contains terms or nested terms that are not separated by a logical operator.

=> 12 and rCTB

24 FILES SEARCHED...

3 FILE DGENE

27 FILES SEARCHED...

52 FILES SEARCHED...

1 FILE SCISEARCH

71 FILES SEARCHED...

2 FILES HAVE ONE OR MORE ANSWERS, 75 FILES SEARCHED IN STNINDEX

L3 QUE L2 AND RCTB

=> 12 and "heat shock protein"

15 FILES SEARCHED...

7 FILE CAPLUS

3 FILE DGENE

27 FILES SEARCHED...

33 FILES SEARCHED...

40 FILES SEARCHED...

48 FILES SEARCHED...

58 FILES SEARCHED...

30 FILE SCISEARCH

1 FILE TOXCENTER

73 FILES SEARCHED...

4 FILES HAVE ONE OR MORE ANSWERS, 75 FILES SEARCHED IN STNINDEX

L4 QUE L2 AND "HEAT SHOCK PROTEIN"

=> d rank

F1 30 SCISEARCH

F2 7 CAPLUS

F3 3 DGENE

F4 1 TOXCENTER

=> file caplus

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FULL ESTIMATED COST

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FILE COVERS 1907 - 25 Mar 2005 VOL 142 ISS 14

FILE LAST UPDATED: 24 Mar 2005 (20050324/ED)

This file contains CAS Registry Numbers for easy and accurate substance identification.

```
=> 12 and "heat shock protein"
      76 "BEHCETS"
      750531 "DISEASE"
      206746 "DISEASES"
      846516 "DISEASE"
            ("DISEASE" OR "DISEASES")
      63 "BEHCET'S DISEASE"
            ("BEHCETS"(W)"DISEASE")
1210216 "HEAT"
      53318 "HEATS"
1223795 "HEAT"
            ("HEAT" OR "HEATS")
      126614 "SHOCK"
      9081 "SHOCKS"
      130715 "SHOCK"
            ("SHOCK" OR "SHOCKS")
1725891 "PROTEIN"
1197591 "PROTEINS"
2003845 "PROTEIN"
            ("PROTEIN" OR "PROTEINS")
      19538 "HEAT SHOCK PROTEIN"
            ("HEAT"(W)"SHOCK"(W)"PROTEIN")
L5      7 L2 AND "HEAT SHOCK PROTEIN"
```

=> d ab bib 1-7

L5 ANSWER 1 OF 7 CAPLUS COPYRIGHT 2005 ACS on STN

AB A review. Autoimmune responses are implicated in the pathogenesis of Behcet's disease (BD). We recently found that the peptide 336-351 of human **heat shock protein 60**, termed Hu-18, provoked vigorous proliferation of T cells from BD patients in Japan, especially those having uveitis. The epitope is specific for BD, because no significant response was detected in patients with RA and normal controls. Characterization of T cell receptor (TCR) usage revealed that T cells expressing particular V beta subfamily were selectively increased in response to Hu-18 stimulation in BD patients. The oligoclonal expansion of Hu-18 specific T cells becomes evident in clin. exacerbation, while it disappears during remission. The same T cell clones were re-expanded in another clin. attack, suggesting the direct involvement of anti-Hu-18 specific T cells in the pathogenesis of BD. The anti-Hu-18 specific T cells were categorized as Th1 cells, because of their cytokine production profile. IL-12 receptor (IL-12R) expressing T cells, which had a high IFN- γ producing potential, were increased in PBL from BD patients with active disease. These data suggest that IL-12/IL-12R system plays a vital role of Th1 polarization during active phase in BD patients. Txk, a member of Tec tyrosine kinase family, is selectively expressed on Th1 and Th0 cells, but not Th2 cells. Txk acts as a transcription factor specific for Th1 T cells. In concordant with Th1 polarization in BD, circulating and tissue infiltrating T cells from the patients expressed abundant Txk protein. Reduction of Txk expression in T cells may lead to the correction of Th1/Th2 imbalance and disease remission in BD. Thus Txk may become a possible therapeutic target in BD.

AN 2004:1061489 CAPLUS

DN 142:153613

TI Autoimmunity in Behcet's disease

AU Suzuki, Noboru; Takeno, Mitsuhiro; Takeba, Yuko; Nagafuchi, Hiroko; Sakane, Tsuyoshi

CS Departments of Immunology and Medicine, St. Marianna University School of

Medicine, Kawasaki, Japan
SO Immunology of Behcet's Disease (2003), 81-86. Editor(s): Zierhut,
Manfred; Ohno, Shigeaki. Publisher: Swets & Zeitlinger B.V., Lisse, Neth.
CODEN: 69GFZ5; ISBN: 90-265-1960-5
DT Conference; General Review
LA English
RE.CNT 22 THERE ARE 22 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 2 OF 7 CAPLUS COPYRIGHT 2005 ACS on STN
AB Behcet's disease (BD) specific peptide (p336-351) was identified within
the human 60 kD **heat shock protein** (HSP60).
Oral p336-351 induced uveitis in rats which was prevented by oral
tolerization with the peptide linked to recombinant cholera toxin B
subunit (CTB). This strategy was adopted in a phase I/II clin. trial by
oral administration of p336-351-CTB, 3 times weekly, followed by gradual
withdrawal of all immunosuppressive drugs used to control the disease in 8
patients with BD. The patients were monitored by clin. and ophthalmol.
examination, as well as extensive immunol. investigations. Oral administration
of p336-351-CTB had no adverse effect and withdrawal of the
immunosuppressive drugs showed no relapse of uveitis in 5 of 8 patients or
5 of 6 selected patients who were free of disease activity prior to
initiating the tolerization regimen. After tolerization was discontinued,
3 of 5 patients remained free of relapsing uveitis for 10-18 mo after
cessation of all treatment. Control of uveitis and extra-ocular
manifestations of BD was associated with a lack of peptide-specific CD4+ T
cell proliferation, a decrease in expression of TH1 type cells (CCR5,
CXCR3), IFN- γ and TNF- α production, CCR7+ T cells and
costimulatory mols. (CD40 and CD28), as compared with an increase in these
parameters in patients in whom uveitis had relapsed. The efficacy of oral
peptide-CTB tolerization will need to be confirmed in a phase III trial,
but this novel strategy in humans might be applicable generally to
autoimmune diseases in which specific antigens have been identified.

AN 2004:640278 CAPLUS
DN 141:364972
TI Oral tolerization with peptide 336-351 linked to cholera toxin B subunit
in preventing relapses of uveitis in Behcet's disease
AU Stanford, M.; Whittall, T.; Bergmeier, L. A.; Lindblad, M.; Lundin, S.;
Shinnick, T.; Mizushima, Y.; Holmgren, J.; Lehner, T.
CS Department of Ophthalmology, Guy's, King's and St. Thomas' School of
Medicine and Dentistry, Guy's Hospital, London, UK
SO Clinical and Experimental Immunology (2004), 137(1), 201-208
CODEN: CEXIAL; ISSN: 0009-9104
PB Blackwell Publishing Ltd.
DT Journal
LA English
RE.CNT 34 THERE ARE 34 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 3 OF 7 CAPLUS COPYRIGHT 2005 ACS on STN
AB A review. Behcet's disease (BD) is a multisystemic inflammatory disorder.
Although the cause and pathogenesis of BD are still unclear, there is
evidence for genetic, immunol. and infectious factors at the onset or in
the course of BD. This review focusses on the functional genomics and
immunol. of BD. HLA-B51 is the major disease susceptibility gene locus in
BD. An increased number of $\gamma\delta$ T cells in the peripheral blood and
in the involved tissues have been reported. However, the T cells at the
sites of inflammation appear to be a phenotypically distinct subset.
There is also a significant $\gamma\delta$ T cell proliferative response to
mycobacterial 65-kDa **heat shock protein**
peptides. Homologous peptides derived from the human 60-kDa **heat**
shock protein were observed in BD patients. There is
evidence that natural killer T cells may also play a role in BD.

AN 2003:819061 CAPLUS
 DN 139:321784
 TI Immunology and functional genomics of Behcet's disease
 AU Zierhut, M.; Mizuki, N.; Ohno, S.; Inoko, H.; Guel, A.; Onoe, K.; Isogai, E.
 CS Department of Ophthalmology, University of Tuebingen, Tuebingen, 72076, Germany
 SO Cellular and Molecular Life Sciences (2003), 60(9), 1903-1922
 CODEN: CMLSFI; ISSN: 1420-682X
 PB Birkhaeuser Verlag
 DT Journal; General Review
 LA English
 RE.CNT 219 THERE ARE 219 CITED REFERENCES AVAILABLE FOR THIS RECORD
 ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 4 OF 7 CAPLUS COPYRIGHT 2005 ACS on STN

AB Although systemic immune reactivity to 65-kD mycobacterial hsp65 (m-hsp65) has been shown previously in Behcet's disease (BD), local immune response was not investigated. We studied anti-m-hsp65 IgG, IgM and IgA antibodies in the serum and cerebrospinal fluid (CSF) of 25 BD patients with cerebral parenchymal involvement (p-NBD), seven BD patients with intracranial hypertension (ih-NBD), eight BD patients without central nervous system (CNS) involvement, 30 patients with multiple sclerosis (MS) and 24 patients with non-inflammatory CNS disorders (NIC). Significantly higher CSF IgG responses were detected in p-NBD patients (ELISA ratio 1.3) compared with NIC (0.7, $P < 0.01$). In p-NBD patients' IgG, IgM or IgA CSF anti-m-hsp65 positivity rate was 48% (12/25); this was significantly higher when compared with MS (3/30; $P < 0.03$) and NIC (3/24; $P < 0.01$). CSF anti-m-hsp65 IgG ratios correlated with the duration of BD ($r = 0.4$, $P < 0.04$) but not with the duration of neurol. involvement. Serum IgM and IgA responses were elevated in ih-NBD, suggesting a different type of involvement than p-NBD. These results implicate an increased local humoral response to m-hsp65 in the CSF of p-NBD patients, which might be related to the pathogenesis of neurol. involvement.

AN 1998:523012 CAPLUS
 DN 129:274568
 TI Humoral immune response to mycobacterial **heat shock protein** (hsp)65 in the cerebrospinal fluid of neuro-Behcet patients
 AU Tasci, B.; Direskeneli, H.; Serdaroglu, P.; Akman-Demir, G.; Eraksoy, M.; Saruhan-Direskeneli, G.
 CS Department of Neurology, Electroneurophysiology Research and Application Centre, Medical Faculty of Istanbul, Istanbul, Turk.
 SO Clinical and Experimental Immunology (1998), 113(1), 100-104
 CODEN: CEXIAL; ISSN: 0009-9104
 PB Blackwell Science Ltd.
 DT Journal
 LA English
 RE.CNT 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS RECORD
 ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 5 OF 7 CAPLUS COPYRIGHT 2005 ACS on STN

AB A review with 53 refs. Behcet's disease is recognized as a systemic inflammatory disease of unknown etiol. The disease has a chronic course with periodic exacerbations and progressive deterioration. Previous reports have shown at least three major pathophysiol. changes in Behcet's disease; excessive functions of neutrophils, vasculitis with endothelial injuries, and autoimmune responses. Many reports suggested that immunol. abnormalities and neutrophil hyperfunction may be involved in the etiol. and the pathophysiol. of this disease. HLA-B51 mols. by themselves may be responsible, in part, for neutrophil hyperfunction in Behcet's disease. T cells in this disease proliferated vigorously in response to a specific

peptide of human **heat shock protein** (hsp) 60 in an antigen-specific fashion. T cells reactive with self-peptides produced Th1-like proinflammatory and/or inflammatory cytokines. This leads to tissue injury, possibly via delayed-type hypersensitivity reaction, macrophage activation, and activation and/or recruitment of neutrophils. These data shed new light on the autoimmune nature of Behcet's disease; mol. mimicry mechanisms may induce and/or exacerbate Behcet's disease by bacterial antigens that have activated T cells which are reactive with self-peptide(s) of hsp. This would lead to pos. selection of autoreactive T cells in this disease.

AN 1998:183177 CAPLUS
DN 128:269272
TI Etiopathology of Behcet's disease: immunological aspects
AU Sakane, Tsuyoshi; Suzuki, Noboru; Nagafuchi, Hiroko
CS Departments of Immunology and Medicine, St. Marianna University School of Medicine, Kawasaki, 216, Japan
SO Yonsei Medical Journal (1997), 38(6), 350-358
CODEN: YOMJA9; ISSN: 0513-5796
PB Yonsei University College of Medicine
DT Journal; General Review
LA English
RE.CNT 53 THERE ARE 53 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 6 OF 7 CAPLUS COPYRIGHT 2005 ACS on STN
AB Peptide 1169-1191 is a major uveitopathogenic determinant of bovine Interphotoreceptor Retinoid Binding Protein (IRBP) in Lewis rats. Previously, we identified two proteins with approx. mol. masses of 72 and 74 kDa and one with a mol. mass of 40 kDa from B cells of naive Lewis rats and EBV-transformed B cells from a human patient with ocular Behcet's disease that bind to bovine IRBP peptide 1169-1191. In this study, we have partially characterized these proteins. The two proteins with mol. masses 72 and 74 kDa belong to the HSP 70 family of proteins and the 40-kDa protein is actin.

AN 1994:698986 CAPLUS
DN 121:298986
TI Characterization of human B cell proteins binding specifically to uveitopathogenic peptide 1169-1191 of bovine IRBP
AU Rengarajan, Kalpana; de Smet, Marc D.; Chader, Gerald J.; Wiggert, Barbara
CS Lab. Retinal Cell and Mol. Biol., Natl. Eye Inst., Bethesda, MD, 20892, USA
SO Biochemical and Biophysical Research Communications (1994), 204(2), 799-806
CODEN: BBRCA9; ISSN: 0006-291X
PB Academic
DT Journal
LA English

L5 ANSWER 7 OF 7 CAPLUS COPYRIGHT 2005 ACS on STN
AB Mycobacterial and homologous human **heat shock protein** T cell peptide epitopes specific for T lymphocytes in Behcet's disease were investigated for their pathogenicity in Lewis rats. The potential pathogenicity of eight peptides and two controls was assessed by administering the peptides in enriched Freund's adjuvant into the footpads of male Lewis rats. Anterior uveitis which is a major manifestation of Behcet's disease was induced with two out of the four mycobacterial and all four homologous human peptides. The most effective peptides inducing iridocyclitis in 64-75% of rats were peptides with amino acids 336-351 and 136-150, derived from the sequence of the human 60 kDa **heat shock protein**. A few of the rats also showed evidence of focal loss of photoreceptors. These results suggest that selected peptides within **heat shock protein** 60 kDa which function as T cell epitopes in Behcet's

disease are capable of inducing uveitis in rats. This supports the view that the peptide T cell determinants may be involved in the pathogenesis of Behcet's disease.

AN 1994:603146 CAPLUS

DN 121:203146

TI **Heat shock protein** peptides reactive in

patients with Behcet's disease are uveitogenic in Lewis rats

AU Stanford, M. R.; Kasp, E.; Whiston, R.; Hasan, A.; Todryk, S.; Shinnick, T.; Mizushima, Y.; Dumonde, Dc.; Zee, R. Van Der; et al.

CS Medical and Dental Schools Guy's, St Thomas' Hospital, London, UK

SO Clinical and Experimental Immunology (1994), 97(2), 226-31

CODEN: CEXIAL; ISSN: 0009-9104

DT Journal

LA Englis

Ref #	Hits	Search Query	DBs	Default Operator	Plurals	Time Stamp
L1	868	recombinant with cholera with toxin B with "sub-unit"	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2005/03/25 17:14
L2	0	recombinant with "cholera toxin B with "sub-unit""	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2005/03/25 17:14
L3	1	recombinant with "cholera toxin B sub-unit"	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2005/03/25 17:15
L4	32	"cholera toxin B sub-unit"	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2005/03/25 17:15
L5	2	"cholera toxin B sub-unit" and "heat shock protein 60"	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2005/03/25 17:16
L6	2148	"Behcet's disease"	US-PGPUB; USPAT; EPO; DERWENT	OR	ON	2005/03/25 17:20